A CONTRIBUTION TO THE STUDY OF CYSTIC KIDNEY.

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The development of cystic kidney is still in many instances obscure, although it has been made the subject of much study, many explanations and theoretical considerations. Individual instances of cystic kidney should always be accurately described and studied so that sufficient reliable and well prepared material can be accumulated from which general deductions could be correctly drawn.

As the matter now stands cysts occurring in the kidney may be divided into the following varieties, for the purpose of orientation:

1. Small cysts found in the kidney of chronic interstitial inflammation and arterio-sclerotic atrophy; these cysts are usually situated in the cortex and are caused by dilatation of the tubules or glomerular capsules on account of compression and obstruction by fibrous tissue. They are not often larger than a big pea. Their contents may consist of retained urine changed by resorption, colloid material from the degeneration of the epithelial lining, blood from hemorrhage into the cavity of the cyst, and these various substances may be present singly or combined, unchanged or, usually, degenerated, giving us variety in color, in consistence, and in microscopic findings. The characteristic epithelial lining may be absent from the larger cysts.

2. Sometimes there is found in an otherwise perfectly healthy kidney a single cyst which may be of the capacity of several litres, projecting prominently from the surface of the organ, or, more frequently, much smaller and then situated either in the cortex or in the medulla; the origin of these cysts is exceedingly difficult to explain; they are obviously retention cysts, and yet the absence of any inflammatory changes in the kidney renders the assignment of any definite cause for the obstruction impossible.[1]

[1] Orth. Lehrb. der speciellen Path. Anat., iv. Lief, Berlin, 1889, p. 141.

3. In the cystic kidney proper—the total cystic degeneration of the kidneys of the Germans and the gros rein polykystique of the French—the entire renal substance, cortex as well as medulla, is crowded with cysts which may be quite large and very small; no distinction between cortical and medullary portions can be pointed out in the very much enlarged kidney. The fluid contents vary much in color, depending on the quantity of pigment, blood, fat, cholestearin, etc., present; chemically the fluid usually contains urea, uric acid, oxalate of lime, albumen and other urinary constituents. Microscopically it is shown that the cyst wall is lined with epithelial cells which often are absent in part or totally in the large cysts; the intercystic tissue is, as a rule, the seat of an advanced chronic interstitial inflammation, but it may contain some quite normal renal parenchyma. Cystic kidney may be unilateral, but oftenest bilateral. It is spoken of as congenital when found in the newborn, in which it may be so voluminous as to constitute a serious obstruction to birth, or death may ensue soon after birth from diaphragmatic impediment, or from renal insufficiency. On the other hand, life may be continued for an indefinite number of years, the cystic kidney remaining latent, the amount of useful renal parenchyma being sufficient to carry on the renal function until gradually advancing interstitial changes, to which the overworked remnants of tissue are predisposed, cause death by uræmia. It is quite plain that the duration of life under these circumstances will depend upon the amount of normal kidney tissue present, and whether the cystic kidney is unilateral or bilateral. All writers suggest the theoretical possibility of congenital cystic changes persisting into middle, or past middle life, and that a considerable number of cystic kidneys in the adult date from birth. The cystic kidney of the adult and old, sometimes spoken of as acquired, resembles in all essentials the congenital, both as to the composition of the cystic fluid and as to the histological structure of the cyst wall and the extra-cystic parenchyma which, however, is almost invariably the seat of chronic interstitial inflammation.

Of sixty-three instances of cystic kidney in the old, collected from medical literature by Lejars,[2] there were in seventeen cases cysts in the liver as well. The relative frequency of contemporaneous cysts in these two organs is so marked that intra-uterine conditions must be looked to for the explanation. It is, of course, true that in many cases the occurrence of kidney and liver cysts in the same person is, so to say, accidental, the hepatic and renal cysts both being easily explained as due to demonstrable local and special causes.

In order to throw all light possible upon the question of etiology

[2] Lejars, Du gros rein polykystique de l'adulte, 1889.

and development it will be well to briefly consider the various theories suggested from time to time as explaining the growth of cystic kidney. A review of the more recent contributions to the literature on this subject at once demonstrates that the explanations offered by the students of the genesis of cystic kidney naturally divide themselves into three sets, namely, the retention, the cyst-adenomatous, and the teratological theories.

1. The theory of retention and dilatation, resulting in the formation of cysts, is based upon the study of a number of cases of cystic kidney in the old as well as in the newborn, in which there was actually demonstrated various causes of obstruction in the uriniferous tubules at the papillæ or elsewhere in their course. Mere mention of the various possible modes of obstruction suggested, but not demonstrated, will be enough; occlusion by the impaction of fibrinous casts, desquamated epithelium, blood, precipitated salts, etc. Virchow[3] assumes a papillary atresia or stenosis due to a fœtal inflammation in order to explain the congenital cystic kidney. Durlach[4] describes an instance in an infant six months old, with large cystic kidneys, the pelvis of which were the seat of a fibrous pyelitis; thick bands of connective tissue passed taperingly up through the columns of Bertini to the surface of the kidney; in the lateral portions of the pyramids was considerable formation of connective tissue and here the development of characteristic retention cysts was beautifully shown.

Complete obliteration or absence of the pelvis is mentioned by Thorn[5], Sutton[6] and others as capable of causing cystic kidney. Thorn describes a kidney removed by Bardenheuer from a man forty-five years old, in which there was a nodular, fibrous pyelitis which attacked by continuity the lateral aspects of the pyramids where there were found many cysts while the central parts of the pyramids were healthy. There were many cysts in the cortex also, but the tissue between the cysts was healthy. Leichtenstern[7] describes a bilateral cystic kidney from a woman forty-eight years old, and selects the name nephropapillitis fibrosa obliterans as descriptive of the lesion demonstrated as the cause of the degeneration; he assumes that the inflammation originated in the coats of the arteriæ rectæ and then passed out among the papillary ducts causing stenosis and retention cysts. Arnold[8]

[3] Wurzburger Verhandlungen, V. p. 461 and Gesammelte Abhandlungen, p. 139-64.

[4] Ueber Entstehung der Cystenniese, 1885.

[5] Beitrag zur Genese der Cystenneisen, 1882.

[6] Sutton, London Lancet, 1887, I. p. 254.

[7] Deutsche med. Wochenschrift, 1884, No. 51.
Ziegler's Beiträge, viii, p. 521.

describes an ascending pyelo-papillitis. Ewald[9] in briefly reviewing the literature on the etiology states that many authors believe that chronic interstitial nephritis with desquamation and impaction of epithelial cells or strangulation of the tubules by connective tissue may cause cystic kidneys in some cases; the peculiarities which would determine the instances in which total cystic degeneration and not merely small cortical cysts should take place are not described. He assumes that in the case described by himself, of a woman, sixty-eight years old, with double polycystic kidney, precipitation of uric acid within the tubules resulted in obstruction and cystic dilatation, because he found in the cysts calculi with uric acid nuclei; he considered the interstitial changes in this case as secondary. Mackenzie[10] reports a case of a woman, sixty-nine years old, with slight double cystic degeneration; in one kidney the cysts contained calculi, in the other there were no stones; he does not consequently place the calculi in any causative relation to the cysts. Smith[11] cites Commandeur's[12] case as one of secondary origin to calculous pyelitis. Lastly it may be stated that the vast majority of cystic kidneys in the old are reported as instances of cystic degeneration accompanying chronic instertitial changes, the relation of the one to the other being left an open question.

2. The cyst-adenomatous theory is based upon the demonstration in cases of cystic kidney of the appearances characteristic of adenomata. In these cases the chronic interstitial changes are usually held to be secondary, although Sabourin[13] concludes his study as follows: Renal cysts in cirrhosis develop from tubules, whose epithelium is reduced to an indifferent state; by fusion cysts are formed, which merit the name of adenoma. Hommey,[14] Lejars,[15] Cornil et Brault,[16] Brigidi and Sevin,[17] Phillippson[18] adopt the same view. Orth[19] calls attention to the fact that in those cases in which the epithelium of the uriniferous tubules is proliferating, and lateral projections form the process is undoubtedly allied to cyst-adenomatous changes. Phillippson saw papillary excresences in the interior of the cysts and solid outgrowths from the uriniferous tubules. The classical adenomas of the kidney are, I believe, usually solid tumors, no matter

[9] Berliner kl. Wochenschr, 1892, No. 1, p. 7, and Deutsch. kl. Wchnschsf., '91, No. 51, p. 1383.

[10] Trans. London Path. Society, xxxix.

[11] Sajous' Annual, 1892, Vol. I, L. 63.

[12] Revue pratique d'obstétrique et d'hygiène de l'enfance, Apr. 5, 1891.

[13] Contribution à l'étude de lo degenerescence kystique des reins et du foie.

[14] Contribution à l'étude anatomique des kystes du rein, 1886.

[15] Loc. cit.

[16] Etudes sur la pathologie des reins.

[17] Lo sperimentale, 1886.

[18] Vichow's Archiv. für Path. Anat. etc., III, page 540, 1889.

[19] Lehrb. der Path. Anat., Lief. IV, Berlin, 1889, p. 144.

what variety it may concern. In the majority of instances of cystic kidney it appears that the epithelium is entirely passive; in adenomatous cysts it would, of course, be active and proliferating, and unless this is demonstrated there seems to be no other criterion by means of which the nature of the process could be established as cyst-adenomatous.

3. Theoretically, the kidney should be the organ of all others, able to furnish instances of the teratoid origin of tumors from persistent embryonic rudiments, but few renal growths have been regarded from this point of view.[20] S. G. Shattock[21] concludes that the congenital cystic kidney is formed by a combination of the meso- and meta-nephros, the remnants of the Wolffian body developing into cysts scattered through the proper renal tissue; the tension upon the intertubular tissue by the enlarging cysts might cause the interstitial changes; the presence of urinary constituents in the cysts does not mean according to Shattock that they originate from the kidney proper, because the meso-nephros in the fishes and the amphibia perform the function of the permanent mammalian kidney. Bland Sutton[22] adopts and advances this view enthusiastically. He says the microscopical appearances of a congenital cystic kidney and of the meso-nephros at its fullest development, the sixth week, agree completely.

Sutton[23] believes that congenital cystic kidney, cystic testicle and parophoron cysts are identical, developmentally, structurally and in their pathology. It is proper to state in connection with this that in many cases of congenital cysts in the kidney other anomalies in development have been found as, for instance, hydrocephalus and hydrencephalocele; horse-shoe or misplaced kidney with congenital cystic change; kidneys without any pelves or ureters; absence of the female genital organs in cases of unilateral cystic kidney. Cases are recorded of the same mother giving birth in succession or interruptedly to several children with cystic kidney.[24] The simultaneous occurrence of cysts in the kidney and in the liver not explainable by local conditions in each organ would naturally be referred, as stated, to intra-uterine conditions in order to account for their origin. This view of Shattock and Sutton is certainly quite fascinating. It is plain however that in the cystic kidney of the old the histological structure in the majority of instances permits no definite conclusion with reference to the possi-

[20] Bell and Johnston, Montreal Medical Journal, February, 1891.

[21] London Pathological Society's Transactions, xxxvii, 1886, p. 287.

[22] London Lancet, 1887, I, page 254 and Gen. Pathology, 1886, 176.

[23] Loc. Cit.

[24] Orth. Loc. Cit.

ble congenital origin of the cysts in persistent meso-nephrotic remnants, because of the advanced changes in the tissue between the cysts and the usual absence of the epithelial lining in the larger cavities. This theory is not mentioned by Ewald.[25]

Conclusions: 1. The common cystic kidney in the old is best explained as due to retention caused by a chronic interstitial inflammation of the pyramids, and the medullary rays especially, as well as of the labyrinthine structure of the cortex. The epithelial lining of the cysts is entirely passive and often absent. There is no epithelial proliferation and new cyst formation demonstrable. A congenital or adenomatous origin for the cysts can only be inferred and not demonstrated.

The etiology of the chronic inflammation is obscure in many cases; in others it may be referred to an ascending pyelo-papillitis from calculi or to the same causes that produce the ordinary chronic interstitial nephritis.

2. Cystic kidneys presenting a proliferating epithelial lining with evidences of new cyst formation by budding from the old cysts or from uniferous tubules are plainly instances of renal cyst-adenomata.

Reasoning by analogy from other organs in the body, occasionally the seat of cystic adenomta, it would be expected that this variety of cystic kidney would most frequently be unilateral.

3. Congenital cystic kidney, without any demonstrable cause for obstruction and urinary retention in the tubules and presenting the structure characteristic of the meso-nephros, as described by Shattock and Sutton, is most reasonably to be regarded as coming from misplaced meso-nephrotic remnants. To look upon all cystic kidneys as congenital cannot be considered proper in view of the instances cited of cysts developing directly as the result of compression of the tubules and the papillæ by fibrous tissue, and cystic adenoma of the kidney can occur at any time of life.

As a typical instance of cystic kidney in the adult I beg to describe the following case:

Unknown woman, about fifty, found in uræmic condition, autopsy showing bilateral large cystic kidney due to a universal chronic interstitial inflammation.

The cystic kidneys to be described were removed from a woman who was picked up in the street by the police, conveyed to the Cook County Hospital, to which she was admitted in an unconscious condition. Judging from her apparel and personal appearance she must have been living in poverty and in unfavorable hygienic surroundings. She was about forty-five or fifty years old. There was deep coma with-

[25] Loc. Cit.

out any convulsive seizures, a foul breath and a furred tongue. The pulse was rapid. Examination of the urine showed this to contain a large amount of albumen, and a diagnosis of uræmic coma was made by Dr. Visser. Death ensued in a few hours.

The post-mortem examination showed quite fair general nourishment. The body bore evidence of imperfect personal hygiene. The great cavities were empty and their lining membranes smooth. The heart showed no valvular lesion, a healthy myocardium with a considerable increase in the thickness of the left ventricular wall. There was a moderate amount of arterio-sclerosis in the aorta. The lungs were œdematous and congested. The spleen, liver and pancreas were quite healthy; there were no cysts in the liver. Both kidneys were greatly enlarged, measuring 18x8x4 cm. in their various diameters;

Fig. 1.—Longitudinal Section of Cystic Kidney—Photograph.

the left weighed 280, the right 297 grammes. The external surface presented a large number of cystic nodules varying in size from that of a pea to a hazel-nut approximately; the cysts were situated on the flat as well as on the convex surfaces of the kidneys, the capsules of which are quite intimately adherent to the cyst walls; there is no normal kidney substance to be made out on the external surface between the cysts which are quite closely aggregated; in places large cysts project from the surface to such an extent as to be almost pedunculated; in other places the cyst barely reaches beyond the surface which is consequently very uneven and nodular, and not altogether unlike a bunch of grapes, The color of the cysts is a light brown, or some modification of brown. On the cut surface there can not be seen any

areas of normal renal substance; all distinction between cortex and medulla has been lost; there are no traces of cortical markings; there are no pyramids to be pointed out, only occasionally can a much changed papillæ be seen projecting into the distorted pelvic cavity. There is no pelvic dilatation, no pressure atrophy of the medullary portion of the kidney, but the cavity of the pelvis is distorted and encroached upon by the crowding into it of cysts from the kidney. The entire cut surface of the kidney is occupied by a vast number of large and small cystic cavities, in equally close apposition near the pelvis as at the circumference, so that the kidney presents a honeycombed appearance throughout. (Fig. 1.) On the cut surface of half of the left kidney two hundred and fifty distinct cysts can easily be counted. The wall of the larger cysts is very thin in places, so that two cavities may be separated from each other by a transparent and

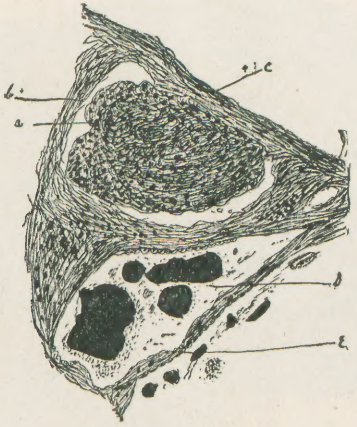

FIG. 2.—*a*. Glomeralus. *b*. Thickened capsule. *c*. Smooth fibrillated wall of large empty cyst. *d*. Cyst with colloid masses; imperfect epithelial lining. *e*. Cyst with colloid and granular masses; epithelial lining barely recognizable. *f*. Small, empty cyst.
Leitz. Obj. 5, Eye-piece III, Tube-length 160 mm. ×150.

delicate membrane; in other places the walls may show transverse ridges and irregularities, undoubtedly the remains of the walls of originally independent cavities which have coalesced by atrophy of the intervening tissue. In the tissue between the larger cysts are many small cavities. The largest cyst measures three cm. in diameter, the smallest are barely distinguishable with the naked eye. In shape the cysts are all somewhat oval, rounded, or oblong; occasionally there is one of angular contour. There is no difference in the shape and in the size of the cysts upon the surface and of those near the pelvis. The cystic contents are all fluid, and quite thin as a rule; in some cysts the fluid was thicker and colloid in appearance; the color

was some shade of brown, and, as a rule, the fluid was slightly opaque. There was no chemical examination made of the fluid further than to demonstrate that it contained albumen and urea. Microscopic examination showed it to hold in suspension the following substances: colloid masses, a few cholestearin tablets, small granular cells with fat drops in their interior in some instances, decolorized red blood-corpuscles, granular detritus, free fat drops.

The pelvic mucous membrane is even, grayish white in color; it does appear a little thickened, but it is not nodular. As before stated, the pelvic cavity is distorded and diminished because cysts crowd into it from the kidney; there does not seem to be much thickening of the

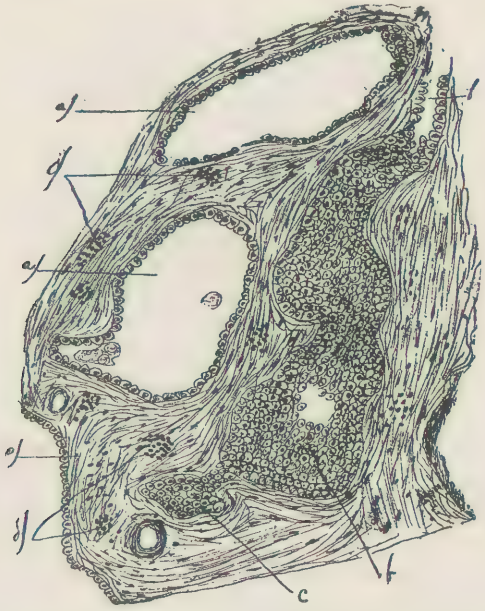

FIG. 3.—*a*. Cysts with epithelial lining. *b*. Tubule with solid outgrowth of cells *c*. Budding mass of *cells*. *d*. Round cell infiltration.
Leitz. Obj. 5, Eye-piece 3, Tube-length 160 mm. × 150.

mucous membrane of the greatly distorted calices; in many places the apex of a pyramid is replaced by a large globular cyst, and nowhere is a typical, normal papilla to be seen.

The ureters, the bladder, and the urethra are quite normal and present no obstruction to the urinary outflow.

Microscopic sections prepared in the usual way—hardened in alcohol, imbedded in parafin or colloidin, stained in hæmotoxylin and eosine, or in carmine—show the following: The microscopic cysts have, as a rule, no epithelial lining, not even a vestige in some cases, in others a row of flattened cells remain; in the

interior are found granular masses, brownish nearly homogeneous oval bodies, small granular cells and detritus; the wall of the large cysts consists of quite dense, fibrillated tissue with slight round cell infiltration; it may contain vessels and capillaries filled with blood. The smaller cysts have, as a rule, a more or less perfect epithelial lining of very short, nucleated columnar cells, but very small cysts may have no lining at all; they contain a debris consisting of amorphous particles and granular cell masses. In a very few places this epithelial lining appears to be proliferating, partly filling the cavity of the tube with cells and sending projections into the surrounding tissue. In Figure 3 is shown a uriniferous tubule with a blind

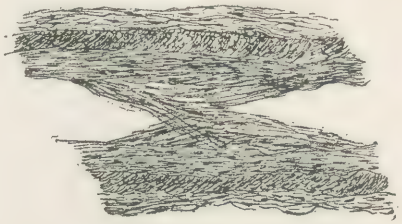

Fig. 4.—Artery in longitudinal section, showing complete obliterating endarteritis. Leitz. Obj. 5, Eye-piece 1, Tube-length 160 mm.×100.

extremity filled with cells and a small cellular mass projecting from its free end to which it is still connected by a rather narrow cellular pedicle. The small cysts are round, or oblong, or kite-shaped. In the large as well as small districts of tissue between the cysts there is an astonishing amount of connective tissue, forming in some places continuous fibrous areas enclosing shrunken, dilated, or entirely collapsed tubules.

The portions of the kidney most extensively involved in the interstitial changes are the medullary portion and the medullary rays in the cortex; the pyramidal part shows the intercystic tissue to be almost entirely fibrous in structure, the cystic cavities being large, oblong, or narrow and long; foci of hemorrhage and of round cell infiltration can be seen in these fibrous districts. The labyrinthine portion of the cortex does not show such an extensive and universal substitution of renal parenchyma with connective tissue and cysts as the medullary rays and the medulla; but the cells lining the tubules are granular, without outline or nucleus, apparently entirely disintegrated. (Fig. 5.) There is nevertheless also a marked increase in the extra-tubular fibrous tissue. In view of these marked changes the number of healthy glomeruli is quite surprising, although the glomerular capsule is quite constantly thickened; in some places there is granular material between the capsule

and the glomerulus, and there are also quite a number of obliterated glomeruli represented by round, dense fibrous masses, and then again one finds healthy glomeruli in continuous, intercystic tracts of connective tissue. The healthy glomeruli appear larger than usual. Sections from the pyramidal part including some of the mucous membranes of the pelvis show, as already stated, an almost continuous dense fibrous tissue, but no such marked involvement of the pelvic membrane as to infer the existence of a fibrous pyelitis which had attacked the papillæ by direct continuity of tissue. Throughout all parts of the kidney the vessels show great thickening of their walls, especially of the intima, amounting in one instance at least to complete obliteration of the lumen of the vessels. (Fig. 4.)

Recapitulation: The cysts with which these kidneys are honeycombed can be plainly traced to dilatation of the straight or collecting uriniferous tubules mainly. The only cause of this dilatation demon-

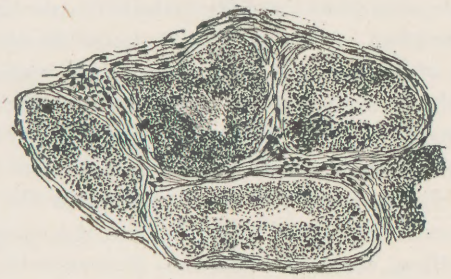

Fig. 5.—Showing degeneration in cells lining the uriniferous tubules; much increase in fibrous tissue and some round cell infiltration. Leitz. Obj. V, Eye-piece 3, Tube-length 160 mm. ×150.

strated by microscopic examination is obliteration in places of the tubules by the contraction of the extra-tubular connective tissue, which is present to an unusually marked extent. The changes in the tissue between the cysts are essentially those of chronic interstitial nephritis. The obviously enlarged glomeruli must be regarded as the seat of a compensatory hypertrophy; and the evidences of epithelial cell proliferation observed here and there in the sections are also to be regarded as the result of compensatory changes in the uriniferous tubules similar in nature to like processes described in the classical chronic interstitial nephritis,[26] as well as in other cases of complete cystic degeneration. This would seem more rational than to look upon the proliferation as adenomatous, according to the view of Sabourin,[27] because it was not possible to demonstrate the formation of new cysts from pre-existing cavities or spaces. To successfully claim a congenital

[26] Orth. loc. cit. page 96.

[27] Loc. cit.

origin of the cysts, regarding the chronic inflammatory changes as secondary, demands precedent in specimens from the adult showing in some way the meso-nephrotic origin of the cysts in a manner that cannot be doubted; no such specimens have as yet been described and recorded. It is consequently the chronic fibrous inflammation found in all parts of the kidneys, especially around the collecting or straight tubules that caused the complete cystic degeneration in this instance of cystic kidneys; the etiology of this inflammation remains obscure; there were no traces of any ascending process that might attack the papillary portions of the pyramids and spread onward into the kidney; it cannot be assumed that the changes are secondary to precipitation of urinary salts in the tubules as in Ewald's case. In fact the only conclusion that can be reached is that it concerns a more marked localization than usual of an ordinary chronic interstitial nephritic process in the medullary portion of the kidney, obstructing the tubules and resulting in peripheral cystic dilatation; the medulla frequently atrophies and suffers from interstitial proliferation in cirrhotic kidney, and in exceptional instances of this disease the process might commence at a very early period in the pyramids before destruction of the secreting parts of the cortex occurred, and by strangulating the straight tubules result in the production of a much greater cystic degeneration than is usually the case. The clinical history of cystic kidney in the adult is essentially that of chronic interstitial nephritis with the occasional addition of bilateral swelling, which is sometimes demonstrable by palpation. Large cystic kidney, due to chronic interstitial nephritis attacking the medulla early or especially, would, as in this case, be expected to occur bilaterally, or there might be a unilateral cystic kidney with the common cirrhotic atrophy of its fellow organ.

The further consideration of the clinical history and diagnosis of cystic kidney in the old can be best elucidated by adding a brief resumè of the articles on this part of the subject by Ewald,[28] Stiller,[29] and Lejars. Ewald's article considers exhaustively the clinical course of cystic kidney, and he finds that the clinical picture is either one of chronic interstitial nephritis, uræmia or complicating inflammations elsewhere ending life, or it remains entirely latent until a rather sudden, fatal uræmia supervenes. Up to the present time cystic kidney has been diagnosed during life only three recorded times: Stiller, verified by the operation, in woman thirty-eight years old, in 1886; Duquet, bilateral cystic kidney in patient fifty-two years old, one year before death, diagnosis based partly on nodularities on the surfaces of the

[28] Loc. Cit.

[29] Berliner kl. Wochenschrift, 1892, No. 215, page 215 and Verhandl. des vii. Congresses für innere Medicin, 1888.

tumors, verified by post-mortem examination; Verneuil, in fifty-three year old patient with complete anuria. Stiller considers the last as a lucky guess rather than a systematically elaborated diagnosis. The following is a brief abstract of the conclusions on diagnosis formulated by Stiller: The topographical diagnosis must be made first. In so doing ovarian tumor will most frequently require exclusion. The topography determined upon, malignant neoplasms come up for consideration. Carcinoma is excluded when the swelling has existed so long or the general health is so good as to be incompatible with presence of this tumor. Youth speaks against carcinoma; childhood does not. Suppurating kidney need not be thought of when the swelling has existed for years, or has been unaccompanied by fever and other symptoms of suppurative nephritis. Nephro-lithiasis, cysto-pyelitis, tuberculosis in other organs may co-exist in rare cases with cystic kidney. Large sacculated kidney, hydro- or pyo-nephrosis or echinococcus cyst, are excluded when the tumor is solid and fluctuation totally or partially absent. Ewald does not agree to this last statement, but maintains that cystic kidney may occasionally give fluctuation. Sudden uræmia or anuria in individuals without history of previous renal disease should direct attention to cystic kidney. If there be bilateral tumor the presumption will be strengthened. In the majority of instances the quality of urine is like that in cirrhotic kidney.

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